



Reevaluating the “deaths of despair” narrative: Racial/ethnic heterogeneity in the trend of psychological distress-related death

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Edited by Bernice Pescosolido, Indiana University Bloomington, Bloomington, IN; received May 7, 2023; accepted November 2, 2023

Despite the significant scientific advancement in deciphering the “deaths of despair” narrative, most relevant studies have focused on drug-, alcohol-, and suicide-related (DAS) deaths. This study directly investigated despair as a determinant of death and the temporal variation and racial heterogeneity among individuals. We used psychological distress (PD) as a proxy for despair and drew data from the US National Health Interview Survey-Linked Mortality Files 1997 to 2014, CDC (Centers for Disease Control and Prevention) Multiple Cause of Death database 1997 to 2014, CDC bridged-race population files 1997 to 2014, Current Population Survey 1997 to 1999, and the American Community Survey 2000 to 2014. We used Cox proportional hazards models to estimate mortality hazard ratios of PD and compared age-standardized PD- and DAS-related mortality rates by race/ethnicity and over time. We found that while Whites had a lower prevalence of PD than Blacks and Hispanics throughout the whole period, they underwent distinctive increases in PD-related death and have had a higher PD-related mortality rate than Blacks and Hispanics since the early 2000s. This was predominantly due to Whites’ relatively high and increasing vulnerability to PD less the prevalence of PD. Furthermore, PD induced a more pervasive mortality consequence than DAS combined for Whites and Blacks. In addition, PD- and DAS-related deaths displayed a concordant trend among Whites but divergent patterns for Blacks and Hispanics. These findings suggest that 1) DAS-related deaths underestimated the mortality consequence of despair for Whites and Blacks but overestimated it for Hispanics; and 2) despair partially contributed to the DAS trend among Whites but probably not for Blacks and Hispanics.

deaths of despair | psychological distress | mortality | drug, alcohol, and suicide | exposure and vulnerability

Ever since Case and Deaton’s (1) *Proceedings of National Academy of Sciences* paper was published, a good amount of research has evolved to decipher the “deaths of despair” narrative. Case and Deaton (1–3) argued that the surge in drug poisoning, suicide, and alcohol-related mortality has contributed to the rising mortality among less-educated working-age White Americans. They used the term deaths of despair as a blanket term to categorize deaths from drugs, alcohol, and suicide (DAS) as these circumstances imply that victims are suffering from psychological distress (PD). Case and Deaton’s work has spurred intense debate about whether despair drives these three causes of death (4–7), whether rising DAS-related death rates are only a White problem (8–10), and whether these three causes are the main contributors to the rising mortality and declining life expectancy for certain demographic groups (9, 11–13) and the widening mortality gap between those with and without higher education (14–16).

Our study focuses on clarifying the trend of despair-induced death and whether DAS-related deaths are driven by despair. Some studies have attempted to address this question by examining the temporal trends in drugs, alcohol, and suicide separately and find the increases in these three causes of death vary by time, geography, and demographic group (17). These findings raise some doubt as to whether these three causes of death share the same underlying etiology (i.e., despair) (9). A counterargument is that despair may manifest differently in different contexts (e.g., as a result of the availability of drugs or suicide prevention programs), which means that one of these causes may be a substitute for the other (18).

Other studies test the drivers of the county variation in drug-related mortality rate and find it is mainly influenced by the supply of legal and illegal drugs than the patterns of economic decline (a possible precursor of despair) (6). A counterargument is that Whites’ sense of despair is not due to economic decline but results from a perceived loss of relative status (7). Other researchers argue that Blacks face historical economic disadvantages and racial discrimination, but have a lower level of despair and have experienced a smaller

Significance

Case and Deaton’s “deaths of despair” narrative has generated significant debate regarding its ability to explain the rising mortality rate and stagnating life expectancy in the United States. This study provides a direct test on despair as a determinant of death, examines the trend of despair-related mortalities, and evaluates how despair contributes to the rising drug-, alcohol-, and suicide (DAS)-related deaths among different racial groups. The findings partially support Case and Deaton’s core argument that distress might have driven the increase in DAS-related deaths among Whites, but highlight the importance of distinguishing between prevalence and vulnerability to distress. They also caution against the limitations of using DAS-related mortalities to understand the pervasive impact of despair or racial differences.

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Author contributions: H.Z. designed research; H.Z. and Y.C. performed research; Y.C. analyzed data; and H.Z. and Y.C. wrote the paper.

The authors declare no competing interest.

This article is a PNAS Direct Submission.

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This article contains supporting information online at <https://www.pnas.org/lookup/suppl/doi:10.1073/pnas.2307656121/-/DCSupplemental>.

Published February 5, 2024.

increase in DAS-related deaths than Whites (9, 19, 20). A possible counterargument is that despite lower exposure to these negative social stressors, Whites may be more susceptible to them, and this vulnerability could have led to greater psychological consequences and mortality.

These prior studies have focused on DAS-related death outcomes and inferred whether the temporal trend or demographic or geographic variation in them is due to despair. This approach, however, has two major limitations. First, deaths related to DAS may not always be indicative of despair itself (5, 6). Secondly, the influence of despair might extend beyond its association with DAS to encompass a broader range of causes of death, such as obesity, cancer, diabetes, and heart diseases. Despair can potentially trigger various unhealthy behaviors, like stress eating (2, 3). Consequently, a sole focus on DAS-related deaths could potentially lead to an underestimation of the broader impact of despair. Therefore, research has yet to surpass these limited death outcomes and directly investigate despair as a determinant of death, while also examining the temporal variation and racial heterogeneity among individuals.

In this study, we use PD as a proxy for despair because both encompass a sense of hopelessness, arise from reaction to stressors in an individual's life, and thus share similar etiologies. By juxtaposing data from various sources, including the US National Health Interview Survey-Linked Mortality (NHIS-LMF), Centers for Disease Control and Prevention (CDC) Multiple Cause of Death (MCD) database, CDC bridged-race population files, Current Population Survey (CPS), and American Community Survey (ACS), we quantitatively assess the death rate and total number of deaths associated with PD. Furthermore, we estimate the temporal trends of these metrics across different racial and ethnic groups. This approach enables a direct evaluation of Case and Deaton's concept of deaths of despair.

Furthermore, we compare the temporal trends in PD-related deaths with DAS-related deaths. This comparison will elucidate (1) whether focusing solely on DAS-related deaths over- or under-estimates the impact of despair on mortality; and (2) whether the trend in DAS-related deaths is driven by despair and how this pattern varies by race and ethnicity. While prior studies have indicated a disproportionate impact of deaths of despair on individuals without a bachelor's (BA) degree (1), our analysis delves deeper into this specific demographic. Given that the pathways leading to certain despair-related deaths (e.g., suicide) may differ by gender (21, 22), we further conduct a gender-specific analysis, aiming to uncover any potential variations in the influence of despair.

Lastly, we employ counterfactual analysis to discern whether the trend in PD-related deaths stems from shifts in PD prevalence or from a changed impact of PD on mortality and to examine how this pattern manifests across different racial groups. This analysis sheds light on existing literature (19, 20) regarding whether the racial heterogeneity in the temporal trends of PD-related mortality reflects varying levels of distress or vulnerability to distress.

Results

Table 1 presents the age-standardized prevalence of PD by race/ethnicity and time periods, both for the whole population and the less-educated group. The percentage of Whites experiencing moderate or severe PD has exhibited a consistent rise, progressing from 10.46 to 11.87% over the period spanning 1997 to 2014. Particularly significant is the magnitude of this increase among Whites without a BA, where the prevalence has risen from 12.52

to 14.89%. The proportion of Blacks with PD increased from 12.77% in 1997 to 2002 to 13.49% in 2003 to 2006, decreased to 12.89% in 2007 to 2010, and rose again to 13.40% in 2011 to 2014. Less-educated Blacks experienced a similar temporal fluctuation. Hispanics generally experienced a declining level of PD from 13.44 to 12.67% between 1997 and 2010 but an increase to 14.22 in 2011 to 2014. During these 17 y, Whites had a 1.41 percentage points increase in the prevalence of PD, which was approximately equal to the increase for Blacks and Hispanics combined (0.63 and 0.78 percentage points increase, respectively). Nonetheless, Whites had a lower prevalence level than Blacks and Hispanics within each period.

Despite a lower prevalence of PD, Whites are more susceptible to it. Table 2 shows the mortality hazard ratio of PD by race/ethnicity and periods (full tables are presented in *SI Appendix, Tables S1 and S2*). Within each period, PD-related mortality hazard ratios were generally higher for Whites than Blacks and Hispanics. For example, in 1997 to 2002, the upper bound estimates show that severe distress was associated with a 114% increase in mortality among Whites, while it was associated with a 44% increase and a 51% increase among Blacks and Hispanics, respectively. Moreover, both moderate and severe PD became increasingly more detrimental over time for Whites, while it became less consequential on mortality for Blacks except during the fourth period, 2011 to 2014, when moderate distress incurred substantially larger mortality consequences than the third period, 2007 to 2010. Among Hispanics, differing levels of distress displayed opposite temporal patterns. Moderate distress-related hazard ratio decreased in the first three periods but increased slightly in the fourth period. In contrast, severe distress-related hazard ratio increased in the first three periods but substantially decreased in the fourth period.

Based on the prevalence and mortality hazard ratio of PD, we constructed population-attributable mortality risk fraction (PAF) and estimated the trend of PD-related deaths from 1997 to 2014. As shown in Fig. 1 (the relevant numbers are displayed in Table 3), in 1997 PD-related mortality rates among Whites were 0.55 (lower bound) – 0.70 (upper bound) per 1,000, lower than Blacks (0.67 to 0.73 per 1,000) but substantially higher than Hispanics (0.26 to 0.29 per 1,000). Over time, Whites underwent a distinctive increase and reached a higher level than Blacks since the early 2000s, which was due to higher PD-related mortality hazard ratios (Table 2), not PD prevalence (Table 1). Blacks and Hispanics displayed a consistent decrease in PD-related mortality rates until 2010 when Blacks experienced a sharp increase, which was probably due to a substantial increase in the mortality hazard ratio of moderate distress (Table 2). In 2014, PD-related mortality rates among Whites were 0.67 to 0.93 per 1,000, higher than both Blacks (0.55 to 0.66 per 1,000), and Hispanics (0.09 to 0.12 per 1,000).

Fig. 1 also displays the comparison between PD-related deaths and DAS-related deaths, which can be used to probe two questions: 1) whether using DAS-related deaths over- or under-estimates the mortality consequence of despair; and 2) whether the trends in these three causes of death are driven by despair. Among Whites and Blacks, PD-related death rates were substantially higher than DAS. For example, the DAS-related death rate in 1997 was 0.32 and 0.34 per 1,000 among Whites and Blacks, respectively, which is about half the PD-related death rate. In contrast, DAS slightly overestimates the mortality consequence of PD among Hispanics. Regarding the second question, among Whites, both DAS- and PD-related mortality rates increased over time, but the former increased at a faster rate, which suggests that the increase in DAS-related mortality might be partially

Table 1. Age-standardized prevalence (%) of PD by race/ethnicity, NHIS, United States, 1997 to 2014

		1997 to 2002	2003 to 2006	2007 to 2010	2011 to 2014
Whole population					
Whites	Sample size	103,155	57,520	47,554	68,192
	None or minimal PD	89.53%	89.08%	88.48%	88.13%
	Moderate PD	7.67%	7.89%	8.33%	8.54%
	Severe PD	2.79%	3.02%	3.19%	3.33%
Blacks	Sample size	20,499	12,566	12,532	16,288
	None or minimal PD	87.23%	86.51%	87.11%	86.59%
	Moderate PD	9.25%	9.65%	9.43%	9.61%
	Severe PD	3.52%	3.84%	3.46%	3.79%
Hispanics	Sample size	23,451	14,830	13,972	18,536
	None or minimal PD	86.56%	87.28%	87.33%	85.79%
	Moderate PD	8.89%	8.95%	8.90%	9.81%
	Severe PD	4.55%	3.77%	3.77%	4.41%
Less-educated population					
Whites	Sample size	74,499	40,042	31,958	44,700
	None or minimal PD	87.48%	86.64%	85.39%	85.11%
	Moderate PD	9.00%	9.43%	10.22%	10.19%
	Severe PD	3.52%	3.93%	4.39%	4.70%
Blacks	Sample size	17,395	10,404	10,193	13,034
	None or minimal PD	86.06%	84.99%	85.28%	84.50%
	Moderate PD	9.98%	10.60%	10.55%	10.90%
	Severe PD	3.96%	4.42%	4.18%	4.60%
Hispanics	Sample size	21,046	13,169	12,114	15,988
	None or minimal PD	85.80%	86.75%	86.56%	84.99%
	Moderate PD	9.31%	9.27%	9.45%	10.21%
	Severe PD	4.89%	3.98%	4.00%	4.80%

Abbreviations: PD, psychological distress.

driven by despair. Among Blacks and Hispanics DAS- and PD-related mortality rates displayed sharply divergent trends. DAS-related death rates were relatively flat until 2010 when they slightly increased, but PD-related mortality rates have consistently decreased over time. These divergent trends indicate that for Blacks and Hispanics, despair was unlikely a primary determinant for the DAS-related mortality trend. The only exception is that after 2010, Blacks experienced a sharp increase in the PD-related mortality rate and a slight increase in the DAS-related mortality rate.

The pattern in the number of deaths mirrors that of the death rate. In 2014, among Whites, PD was linked to a substantial 100,550 to 137,760 deaths, a notably higher figure than deaths related to DAS (83,319). Similarly, within the Black population, PD contributed to 14,850 to 17,721 deaths, surpassing the count of deaths attributed to DAS (8,478). In contrast, among Hispanics, DAS-related deaths totaled 9,541, exceeding the 2,923 to 3,813 deaths associated with PD.

Within the less-educated demographic, a consistent racial and temporal pattern emerges (*SI Appendix, Fig. S1 and Tables S3–S5*). Among individuals without a BA degree, there was a distinct rise in PD-related mortality rates for Whites from 1997 to 2014, surpassing the rates observed among both Blacks and Hispanics without a BA over the entire duration. In 2014, PD-related death rates stood at 1.03 to 1.42 per 1,000 for Whites, notably exceeding the figures of 0.70 to 0.85 per 1,000 for Blacks and 0.10 to 0.13 per 1,000 for Hispanics (refer to Table 3).

We extend our analysis to explore gender-specific racial differences in both PD- and DAS-related death rates (*SI Appendix,*

Tables S6–S8). Notably, among White women, the increase in PD-related mortality rate from 1997 to 2014 is even more pronounced than that observed among White men. The rates have risen from 0.50 to 0.66 per 1,000 in 1997 to 0.67 to 0.93 per 1,000 in 2014 for White women, compared to 0.60 to 0.74 per 1,000 to 0.65 to 0.89 per 1,000 for White men. Furthermore, the difference in PD- and DAS-related mortality rates is substantially larger for White and Black women compared to their male counterparts. In essence, this implies that relying solely on DAS-related deaths results in a greater underestimation of the mortality impact of despair among White and Black women than among their male counterparts. Conversely, the use of DAS overestimates the mortality consequences of PD, particularly among Hispanic men as opposed to Hispanic women.

What contributes to the differential racial trends in PD-related death rates? The results from simulation analyses displayed in Fig. 2 suggest that among Whites, the rising PD-related death rate was primarily caused by the increasing vulnerability to PD although the rising prevalence of PD also made a sizable contribution. If the mortality hazard ratio of PD was fixed at the 1997 to 2002 level, the PD-related mortality rate would have only increased by 6% instead of 33% from 1997 to 2014. If the prevalence of PD was fixed at the 1997 to 2002 level, this rate would have increased by 18%. Among Black and Hispanics, the trend of PD-related mortality rate was almost completely due to the decreasing vulnerability to PD. If the mortality hazard ratio of PD was fixed at the 1997 to 2002 level, the PD-related death rate would have only slightly declined (Blacks) or flattened (Hispanics). These counterfactual analyses suggest that the heterogeneous racial

Table 2. PD-related mortality hazard ratio [95% CI] from Cox hazard models by race/ethnicity in four periods, NHIS-LMF, United States, 1997 to 2014

		1997 to 2002	2003 to 2006	2007 to 2010	2011 to 2014
Whites			Lower bound*		
	Moderate PD	1.435 [1.286,1.601]	1.493 [1.351,1.649]	1.534 [1.402,1.678]	1.522 [1.400,1.653]
	Severe PD	1.838 [1.581,2.138]	1.777 [1.531,2.062]	1.826 [1.602,2.081]	1.938 [1.739,2.160]
			Upper bound†		
	Moderate PD	1.541 [1.380,1.720]	1.662 [1.508,1.832]	1.691 [1.547,1.850]	1.727 [1.594,1.871]
	Severe PD	2.137 [1.837,2.486]	2.125 [1.837,2.457]	2.204 [1.933,2.513]	2.351 [2.106,2.625]
	Observations	371,022	556,001	732,104	938,573
			Lower bound*		
Blacks	Moderate PD	1.535 [1.234,1.909]	1.253 [1.022,1.536]	1.097 [0.912,1.319]	1.488 [1.272,1.740]
	Severe PD	1.38 [0.958,1.988]	1.597 [1.208,2.110]	1.566 [1.203,2.040]	1.413 [1.115,1.789]
			Upper bound†		
	Moderate PD	1.577 [1.267,1.962]	1.329 [1.081,1.635]	1.145 [0.951,1.378]	1.567 [1.339,1.834]
	Severe PD	1.442 [1.004,2.069]	1.853 [1.391,2.467]	1.716 [1.321,2.228]	1.556 [1.227,1.972]
	Observations	74,313	114,634	161,595	218,043
			Lower bound*		
	Hispanics	Moderate PD	1.473 [1.109,1.956]	1.339 [1.044,1.716]	1.099 [0.879,1.374]
Severe PD		1.415 [0.961,2.084]	1.426 [1.036,1.963]	1.534 [1.169,2.012]	1.182 [0.936,1.492]
			Upper bound†		
Moderate PD		1.528 [1.144,2.041]	1.374 [1.070,1.764]	1.141 [0.909,1.432]	1.183 [0.994,1.408]
Severe PD		1.507 [1.015,2.238]	1.532 [1.109,2.115]	1.67 [1.278,2.181]	1.284 [1.013,1.627]
Observations		85,639	140,366	198,337	267,431

For each model, None or minimal PD is the reference group.

*Lower bound controlling for gender, college education, poverty income ratio, employment status, marital status, # of children, pain, smoking, drinking, obesity, and year.

†Upper bound controlling for gender, college education, poverty income ratio, employment status, marital status, # of children, pain, and year.

trends in PD-related mortality rate were primarily a result of differential trends in susceptibility to PD not the prevalence of PD.

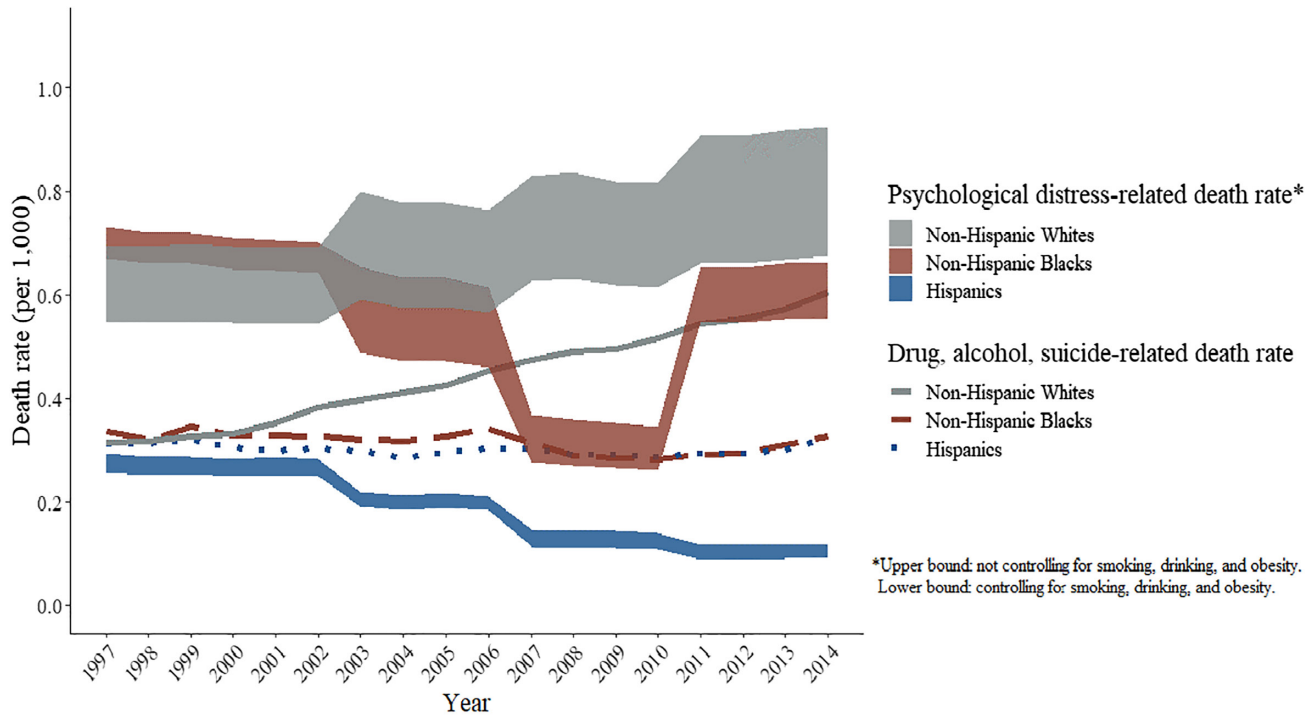
Discussion

Our main aim was to investigate racial/ethnic heterogeneity in the temporal pattern of PD-related mortality and compare it to DAS-related mortality. This approach allowed us to directly evaluate Case and Deaton's deaths of despair narrative and infer whether DAS-related deaths over- or under-estimate the mortality consequence of despair and whether the temporal trend in DAS-related deaths is driven by despair. Our findings reveal distinctive and continuous increases in PD-related deaths among Whites, with their rates surpassing those of Blacks and Hispanics since the early 2000s. This phenomenon is primarily attributed to Whites' heightened and escalating vulnerability to PD. While the growing prevalence of PD also contributed, it is noteworthy that Whites exhibited a lower prevalence of PD compared to

Blacks and Hispanics across the period from 1997 to 2014. These results lend partial support to Case and Deaton's deaths of despair narrative. On the one hand, this narrative correctly portrays the worsening *trend* in PD and its increasing impact on mortality among Whites. However, on the other hand, this narrative might be inaccurate as it could potentially mask the fact that Blacks and Hispanics experience higher *levels* of PD than Whites.

Regarding the comparison between PD and DAS, DAS-related deaths under-estimate the mortality consequence of despair for Whites and Blacks but overestimate it for Hispanics. In other words, despair-induced deaths are not completely captured by DAS for Whites and Blacks. This supports the notion proposed by Case and Deaton (2, 3) that the mortality consequences of despair extend more broadly beyond DAS, at least for Whites and Blacks. However, these findings also imply that relying solely on DAS is insufficient in comprehending the pervasive influence of despair or the existing racial disparities.

A Age-standardized mortality rate



B Number of deaths

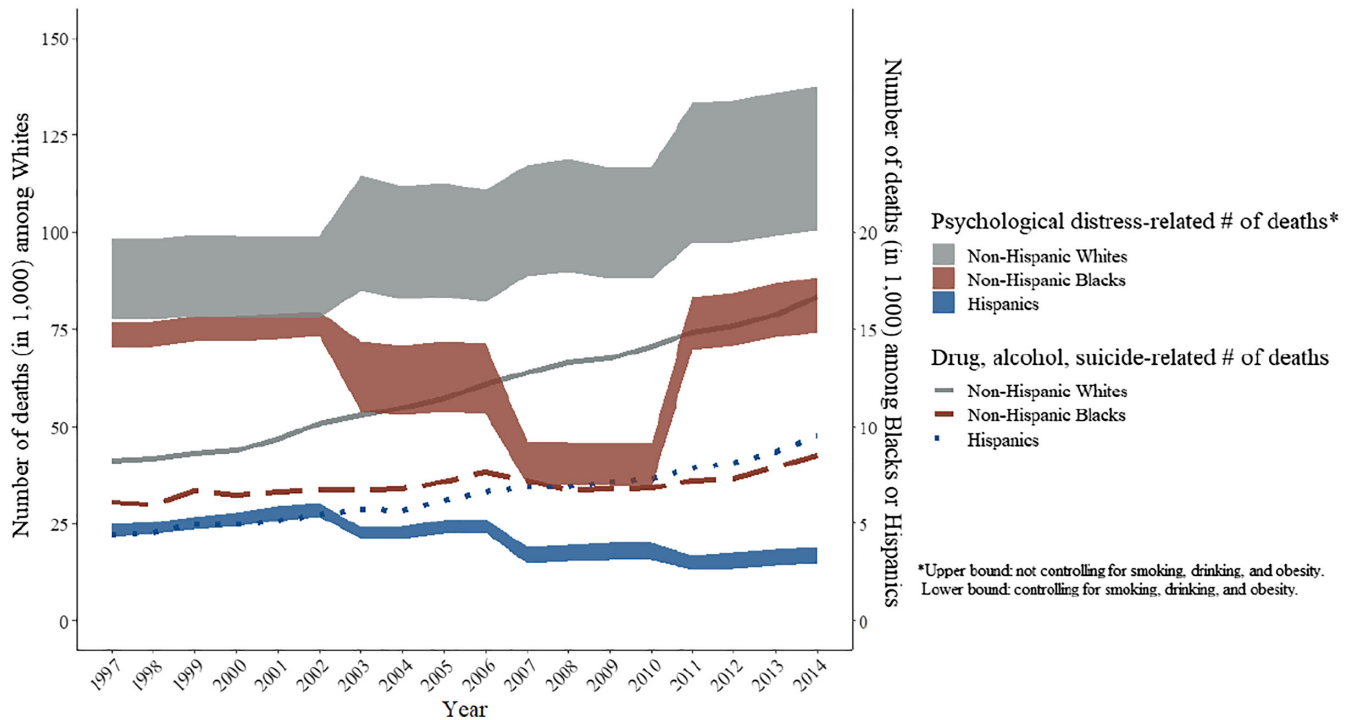


Fig. 1. Age-standardized mortality rate and number of deaths by race/ethnicity, United States, 1997 to 2014. (A) Age-standardized mortality rate. (B) Number of deaths.

Regarding the temporal trend, increasing DAS-related mortality rates over time among Whites may be partially driven by increasing vulnerability to despair although rising despair levels play a role as well. In contrast, Blacks and Hispanics became less susceptible to despair over time, which led to decreasing PD-related mortality rates before 2010. DAS-related mortality rates, however, were relatively flat during this period, which implies that these temporal patterns

probably were not driven by despair. However, the slight increase in DAS-related mortality among Blacks after 2010 is concordant with the significant surge in PD-related mortality rates, which may suggest that PD began to affect DAS, although divergent trends before 2010 may cast doubt on PD's causal role on DAS after 2010.

These findings carry significant implications for the ongoing debate within suicidology, which critiques the White-centered

Table 3. Age-standardized death rates (per 1,000) and number of deaths for PD-related death and DAS-related death in Fig. 1 and *SI Appendix, Fig. S1*, United States, 1997 and 2014

	Total population (Fig. 1)		
	White	Black	Hispanic
	Age-standardized death rates (per 1,000)		
PD			
1997	0.55 to 0.70	0.67 to 0.73	0.26 to 0.29
2014	0.67 to 0.93	0.55 to 0.66	0.09 to 0.12
DAS			
1997	0.32	0.34	0.31
2014	0.61	0.33	0.32
	Number of deaths		
PD			
1997	77,409 to 98,301	14,058 to 15,325	4,351 to 4,955
2014	100,550 to 137,760	14,850 to 17,721	2,923 to 3,813
DAS			
1997	41,130	6,118	4,433
2014	83,319	8,478	9,541
	Less-educated population (<i>SI Appendix, Fig. S1</i>)		
	White	Black	Hispanic
	Age-standardized death rates (per 1,000)		
PD			
1997	0.82 to 1.03	0.78 to 0.87	0.29 to 0.32
2014	1.03 to 1.42	0.70 to 0.85	0.10 to 0.13
DAS			
1997	0.38	0.38	0.33
2014	0.81	0.39	0.35
	Number of deaths		
PD			
1997	80,002 to 100,309	13,488 to 15,063	4,243 to 4,816
2014	96,628 to 133,275	14,492 to 17,512	2,709 to 3,542
DAS			
1997	34,929	5,737	4,207
2014	69,994	7,792	8,956

Abbreviations: PD, psychological distress; DAS, drug, alcohol, and suicide.

approach to comprehending the underlying causes of suicide (23). Our findings suggest that while despair might be a driving factor behind the *trend* of DAS (including suicide) among Whites, the same might not hold true for Blacks and Hispanics. This underscores the importance of refraining from assuming despair as the sole catalyst for DAS and emphasizes the necessity of adopting an intersectional approach rather than a White-centered one. Furthermore, these findings advise against employing deaths of despair as a sweeping term to categorize fatalities resulting from drugs, alcohol, and suicide. They emphasize that the pathways leading to these three causes of death could differ significantly among different demographic groups. As a result, in order to comprehend deaths stemming from despair, it is crucial to view despair as a determinant rather than solely relying on the outcomes of death.

Our findings raise several important questions that require further studies, but we provide some tentative explanations here. First, why are Whites more susceptible to despair despite a lower level of despair than Blacks and Hispanics? A similar puzzle in the literature is that despite a higher level of PD (24, 25), Blacks and Hispanics have a lower level of major depressive disorder (26–31). This has

been attributed to statistical artefacts resulting from undercounting and undertreatment of psychiatric disorders among non-Whites (32, 33) and some protective factors are more prevalent among non-Whites, such as religiosity and strong social support (29, 34–36). These protective factors may temper the impact of despair.

Second, why is the despair level continuously increasing over time among Whites but not Blacks and Hispanics? Relatedly, why are Whites becoming more susceptible to despair over time but Blacks and Hispanics are not? Both patterns among Whites may be concomitant with shrinking manufacturing employment (3, 37, 38), the relative loss of status (7, 39, 40), a steep waning in religious participation (41), and a more recent decline in marriage (42, 43) that are particularly experienced by Whites. However, how much these factors causally account for the racial difference in the trend of exposure and vulnerability to PD requires further investigation.

This study has several limitations. First, we use PD as a proxy for despair as both encompass a sense of hopelessness. Arguably, other possible proxies might be utilized, such as psychological pain, which may be a closer precursor to certain deaths (e.g., suicide)

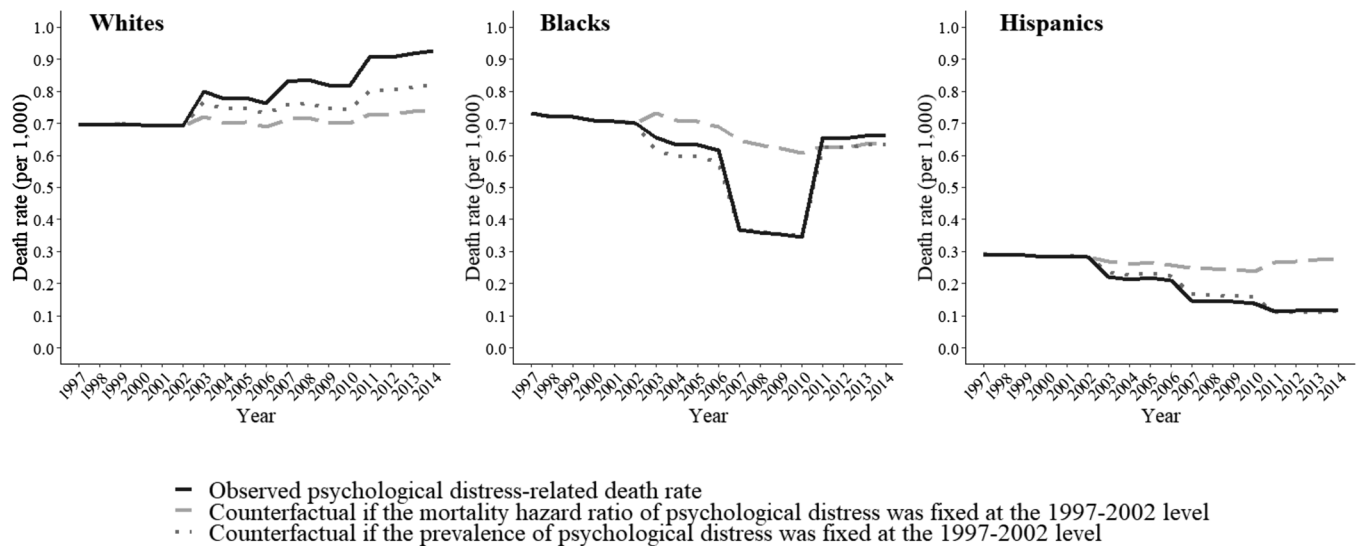


Fig. 2. Counterfactual trend in PD-related death rate if the prevalence or mortality hazard ratio of PD was fixed at the 1997 to 2002 level, NHIS-LMF, United States, 1997 to 2014.

than PD. However, this approach would necessitate appropriate data to determine the temporal trend in the association between these proxies and mortality, as well as racial differences in these temporal trends. Recent studies have indicated a rising prevalence of physical or chronic pain (44, 45) and proposed that pain could serve as a pathway to suicide, as well as to drug and alcohol addiction (1). We conducted analyses using pain as a proxy for despair. However, our findings indicate that pain is not significantly associated with mortality hazard in most analyses (*SI Appendix, Table S9*), and the pain-related death rate and number of deaths are found to be relatively moderate (*SI Appendix, Table S10*). Nonetheless, the pain-related mortality rate increases among Whites while either stagnating or decreasing among Blacks and Hispanics, a pattern consistent with the PD-related mortality rate.

Second, NHIS does not have longitudinal data on PD. To curtail this limitation, we restricted the mortality follow-up window to 5 y and re-estimated PD-related mortality by race and time periods. The overall racial and temporal patterns were similar although the values of PD-related mortality hazard ratio slightly varied. Third, the NHIS population exhibits better health and longevity compared to the general population (46) because the survey excludes institutionalized individuals (47). As a result, the actual mortality hazard associated with PD might be higher if individuals with poorer health were included in the analysis. The upper bound of our estimates is at risk of underestimation. Fourth, due to the observational nature of NHIS data, we should be cautious about drawing a causal inference. Nonetheless, we have controlled a series of demographic, socioeconomic, physical, and bio-behavioral factors to obtain the lower-bound estimates of the relationship between PD and mortality.

Fifth, because NHIS cause-specific death data are restricted after 2004, we are unable to analyze the correlation between PD and cause-specific deaths over time and by racial/ethnic groups. Arguably, the impact of despair on mortality extends beyond any single cause of death. Notwithstanding, such analysis could provide additional insights into the diverse racial patterns and temporal variations underlying the mechanisms through which despair leads to deaths. Our preliminary analysis based on data before 2004 finds that PD is linked to various causes of death, including cardiovascular diseases, cancer, respiratory diseases, accidents, Alzheimer's disease, and deaths related to DAS. Moreover, PD

holds more substantial implications for Whites in most of these causes of death. However, it is important to recognize that results concerning DAS may not be entirely reliable due to the limited number of deaths from these specific causes. Future research should continue pursuing this research idea and investigate racial differences in the temporal trends of the link between PD and various causes of death.

Remarkably distinct trends in mortality associated with despair have surfaced among Whites, Blacks, and Hispanics over the past two decades. This divergence is primarily attributed to differing trends in vulnerability to despair rather than disparate trends in the prevalence of despair itself. It is evident that comprehending the pervasive impact of despair or the existing racial disparities cannot be adequately achieved solely through an analysis of drugs, alcohol, and suicide-related deaths. Future research should extend beyond these specific causes of death and delve deeper into identifying the underlying factors that propel the unique susceptibility trends to despair across various racial/ethnic groups.

Materials and Methods

Data. We used data from NHIS-LMF 1997 to 2014, CDC MCD database 1997 to 2014, CDC bridged-race population 1997 to 2014, CPS 1997 to 1999, and ACS 2000 to 2014. NHIS-LMF files were obtained from the Integrated Public Use Microdata Series (IPUMS) (48). The NHIS is an annual cross-sectional, multistage probability sample survey of the non-institutionalized US civilian population. It is linked to death records in the National Death Index through probabilistic record-matching methods, which use 13 criteria to ascertain the vital status of each respondent. At the time of the study, the death records were updated to 31 December 2014. *SI Appendix, 1* shows the flowchart of the study sample. Of the 529,363 individuals with eligible mortality records (95.8% of the sample), we excluded respondents in other race/ethnic categories ($n = 27,050$), with missing data on education ($n = 2,890$), PD ($n = 7,439$), marital status ($n = 1,250$), employment status ($n = 190$), body mass index (BMI) ($n = 15,943$), drinking status ($n = 3,650$), smoking status ($n = 396$), and physical pain measures ($n = 590$). The sample included does not display a substantial difference in the demographic characteristics and PD level from those excluded. After reshaping the data into the long format, we restricted observations to person-years from respondents ages 25 to 84 (dropping 60,870 individuals) because most respondents had finished their educations by age 25, the age at the interview was top coded at 85, and mortality matches were not very reliable among individuals ages 85 and older. The final analytical sample contains 409,095 individuals (276,421 non-Hispanic Whites, 61,885 non-Hispanic Blacks, and 70,789 Hispanics), 3,858,058

observations, and 40,437 deaths. *SI Appendix, Table S11* displays the sample size and descriptive statistics. We used this dataset to estimate the prevalence and mortality hazard of PD.

The MCD database contains the decedents' age, gender, race, ethnicity, education, and cause of death. Missing data for education (on average 5.8% of cases from 1997 to 2014) were imputed using age, gender, race, ethnicity, and cause of death information following prior studies (15, 16, 49). CDC bridged-race population files contain US population size by age, gender, race, and ethnicity, which are based on estimates produced by the US Census Bureau. In order to categorize the population by education level, the CPS 1997 to 1999 and ACS 2000 to 2014 were used, which were obtained from IPUMS. Both databases use nationally representative US household samples and are administered by the US Census Bureau. The number of DAS-related deaths was estimated from MCD while midyear population estimates were obtained from CDC bridged-race population files, CPS, or ACS. To be comparable to NHIS-LMF, these four datasets were constrained to ages 25 to 84. Despite the risk of a potential mismatch between education reported in the CPS or ACS and that recorded on the death certificate (50), many studies have used these data to construct education-specific mortality rates (1, 2, 15, 16, 51–53) and found similar education and mortality trends based on NHIS (46). This further substantiates the validity of comparing the mortality trend based on administrative data and survey data.

Measures. Race/ethnicity consisted of three groups: non-Hispanic Whites, non-Hispanic Blacks, and Hispanics. Education was coded into a dichotomous variable: a completed college degree vs. less than a college degree. This dichotomy represents the major pattern of educational inequalities in PD and mortality (3). In the MCD database, causes of death were recorded using the 9th Revision (1997 to 1998) and the 10th Revision (1999 to 2014) of the International Classification of Diseases. *SI Appendix, 2* shows the codes for DAS-related deaths.

In the NHIS-LMF data, we measured PD using the Kessler 6 scale, a six-item instrument that asks respondents how often during the preceding 30 d, from none of the time (scored as 0) to all of the time (scored as 4), they felt sad, nervous, restless, hopeless, worthless, or that everything was an effort. We categorized the total range (0 to 24) into three groups: none or minimally distressed (range 0 to 6), somewhat distressed (7 to 12), and severely distressed (13 to 24). This categorization is well established in the literature (54–56). Using slightly different cutoff points (e.g., 7 to 14, 15 to 24) produced qualitatively the same results.

Marital status was categorized as married, divorced or separated, widowed, and never married. Poverty income ratio was the ratio of total household income to a year-specific federal poverty threshold. It was categorized into four groups: below the poverty line (less than 1), middle (1 to 1.99), high (2 or more), and unknown. Employment status included unemployed, employed, and not in the labor force. The number of children was categorized as none, one, two, or more.

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Pain summary index was based on the number of pains during the past 3 mo in the following five sites: lower back, leg, neck, face/jaw, and head (severe headaches or migraines). Smoking status was categorized as never smoking, former smoker, and current smoker. Drinking status consisted of never drinking, former drinker, current drinker, and binge drinker. Binge drinking was defined as more than 60 d of five or more drinks per day in the past year (57, 58). BMI had 5 categories: underweight (BMI < 18.5), normal weight (18.5 ≤ BMI < 25), overweight (25 ≤ BMI < 30), obesity I (30 ≤ BMI < 35), and obesity II/III (BMI ≥ 35).

Analysis. We used Cox proportional hazards models adjusted for sampling weight to estimate mortality hazard ratios of PD by racial/ethnic groups and periods (1997 to 2002, 2003 to 2006, 2007 to 2010, and 2011 to 2014). Age was used as the time metric, with subjects entering the risk set at the age at the time of the survey and exiting at the event (death or censoring age). After obtaining the hazard ratios for PD, we calculated the PAF to estimate the proportion of deaths in the population due to PD for each racial group and period (*SI Appendix, 3*). We used the mortality hazard ratio with and without adjusting for bio-behavioral factors (smoking, drinking, and BMI) to construct the lower bound and upper bound of PAF because they can be the mechanisms linking PD to mortality. To eliminate the impact of differential age compositions across racial groups and periods, we used the age-standardized prevalence of PD to calculate PAF. We standardized the prevalence to the age distribution of the 2000 US population by 5-y age groups (25 to 29, 30 to 34, ..., 80 to 84) using data from CDC bridged-race population. PD-related number of deaths was obtained by multiplying PAF by the total number of deaths estimated from MCD. PD-related mortality rate was obtained by dividing the related number of deaths by population size estimated from CDC bridged-race population.

With regard to DAS-related death rate, the number of deaths was obtained from MCD data, and midyear population estimates were obtained from CDC bridged-race population. Mortality rates were categorized by 5-y age groups from ages 25 to 84 for each racial/ethnic group. Similar to PD-related death rates, DAS-related death rates were age-standardized using the 2000 age structure by 5-y age groups. We repeated all the analyses for the less-educated group using the same approaches and employed population estimates from CPS and ACS.

We further used simulation to evaluate the contribution of changing prevalence of PD or changing mortality hazard ratio of PD to its related mortality rate over time by fixing them at the 1997 to 2002 level. Using the simulated PAF, we obtained the counterfactual trend of PD-related mortality rate over time. These analyses were also conducted within each racial group.

Data, Materials, and Software Availability. Anonymized data have been deposited in OSF (DOI: [10.17605/OSF.IO/RTZC2](https://doi.org/10.17605/OSF.IO/RTZC2)) (59).

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